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ABSTRACT

Developmental models currently used by child behavioral toxicologists and teratologists are inadequate to address current issues in these fields. Both child behavioral teratology and toxicology scientifically study the impact of exposure to toxic agents on behavior development: teratology focuses on prenatal exposure and postnatal behavior development; toxicology on postnatal exposure and subsequent behavioral development. In general, the field of behavioral toxicology has underestimated, dismissed, or poorly evaluated the potential impact of psychosocial variables in determining an infant's or child's vulnerability to toxicants. Given the complexity of developmental processes, behavioral toxicologists and teratologists must adopt developmental models more complex than the "main effects" model currently in use. The more realistic interactive model, while better than the main effects model, is also inadequate since its elements are essentially static. Developmental toxicology must adopt a transactional model, or one that takes into account the ongoing transactions between individual biological and behavioral factors and the environmental contexts in which they operate. (An outline of a test battery formulated to link lead exposure to developmental delays is discussed in terms of five aspects that make it capable of addressing issues generated by the transactional model as applied to child behavioral toxicology.) (RH)

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Research Models in Developmental Behavioral Toxicology

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We come to two major conclusions in this paper: (1) the developmental models currently used by child behavioral toxicologists and teratologists are inadequate in relation to the issues that are now being addressed in these fields, and (2) the field of behavioral toxicology has underestimated, dismissed, or poorly evaluated the potential impact of psychosocial variables in determining an infant's or child's vulnerability to toxicants.

The fields of human behavioral toxicology and teratology are relatively new. Experimental and clinical psychologists have only recently joined biochemists, neurobiologists, and epidemiologists in the examination of the effects of xenobiotics on human growth and development. Toxicology and teratology have turned to the behavioral sciences because of a recognition that delays or aberrations in psychological development may serve as sensitive indicators of a toxic action, particularly in the central nervous system (Weiss & Spyker, 1974; Spyker, 1975; Vorhees & Butcher, 1982).

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Even more recent is the emerging "specialization" of the behavioral toxicology and teratology of childhood (Marcus & Hans, 1982; Fein, Schwartz, Jacobson, & Jacobson, In Press). Society is becoming increasingly concerned with the impact of industrial toxicants and licit and illicit drugs on children's mental development, and behavioral scientists are beginning to respond with programs of research and intervention.

By way of definition, child behavioral teratology is the scientific study of the impact of prenatal exposure to toxic agents on postnatal behavioral development, while child behavioral toxicology is the scientific study of the impact of postnatal exposure to toxicants on behavioral development. There is naturally a great deal of overlap between these two areas of concern. For example, mercury, lead, and PCB's have been studied as both behavioral teratogens and postnatal behavioral toxicants. Also, both child behavioral teratologists and toxicologists are concerned with behavioral development, taking both areas into the field of developmental psychology.

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However, developmental psychology is a specialization in itself. There exists a host of factors which contribute to developmental processes and we are just beginning to understand how they work together. Such factors as genetics, parenting practices, and medical history (to focus on only global variables) not only enter singly into an individual's developmental trajectory, but also interact in potentially complex ways.

Developmental psychologists and behavioral toxicologists have known for some time that untangling the contribution these variables make to behavioral development is almost impossible using purely retrospective research designs--thus, longitudinal or quasi-longitudinal designs have become the research strategies of choice. However, as essential as they are, longitudinal designs are not a panacea. In an attempt to understand the complexities of human behavioral development, psychologists have had to adopt complex, yet powerful developmental models as an aid in the formulation of hypotheses, choice of measures, and methods of statistical analysis. We feel that behavioral toxicologists and teratologists must now do the same.

Unfortunately, most if not all reported studies of the behavioral toxicology of childhood adhere to a simple Main Effects Model of development. In this model, biological and environmental variables are felt to exert influences on behavioral development which are independent of each other. In figure 1, the toxic effect of lead (Pb) is felt to exert itself irrespective of other factors which influence development, the most obvious of which are the child's constitutional endowment, state of health, and psychosocial milieu.

In applying the main effects model, some investigators have recognized the necessity to control for the impact of psychosocial factors. However, this has typically been done either by employing status measures of such variables (e.g., ratings of socioeconomic class), and then statistically partialling out their effects, or by sampling from a "socioeconomically homogeneous" stratum. However, this application fails to account for the potentially dynamic interaction between biomedical and social factors--that is, the liklihood that infants and children within various sociocultural environments may be differentially vulnerable to the functional effects of a toxicant.

Since such research typically employs ratings of social class, a critical issue that emerges is whether or not such status measures are sensitive to variations in the caretaking environments of children in toxicological studies. Figure 2 presents caretaking data on a group of infants who occupy Hollingshead's lowest social position category. The caretaking measure is Caldwell and Bradley's (1979) Home Observation for Measurement of the Environment, a direct observational measure of the infant's social and non-social developmental milieu. As shown, a great deal of intraclass variability exists in the quality of caretaking as indexed by the home measure. These data seem to indicate that toxicological studies would fail to control for environmental differences by sampling from a socieconomically homogeneous stratum, or statistically by entering socioeconomic ratings into an analysis of covariance. This problem is particularly critical since the Home Observation for Measurement of the Environment has been shown to predict later intellectual achievement better than status indices of social class.

Returning to our discussion of developmental models, the Interactive Developmental Model was fostered by psychologists to explain how biological and environmental variables may exert cumulative deleterious, positive, or ameliorating influences on behavioral development. The interactive model has been particularly useful to investigators studying the developmental sequelae of various perinatal risk factors, since such work has shown that it is the interaction between constitutional and experiential factors which determines later developmental status (Broman, Nichols, & Kennedy, 1975). Figure 3 illustrates a hypothetical example from toxicology. As shown, the deleterious effects of lead toxicity might be exacerbated in an infant affected by adverse biological factors (e.g., prematurity), but otherwise ameliorated or compensated for in infants who are constitutionally sound

and/or developing in a positive social environment. To summarize, the interactive developmental model allows one to uncover toxic effects as they are more likely to occur in the real world where there is an interplay between social processes, biological factors, and toxicants in behavioral development.

The interactive model is clearly an improvement over the main effects approach. However, even this more advanced approach is inadequate since its elements are essentilly static. The model assumes that the pertinent characteristics of the child and his environment combine in an additive manner and remain stable over time. But research in child development has indicated that this is clearly not the case. There is ample evidence that the biologicaland behavioral characteristics of the child influence the way in which the environment responds to the infant and that, in turn, the quality of social responses given to the infant changes the child's behavior (Sameroff, 1978).

Developmental toxicology must therefore adopt a model which takes into account the ongoing transactions between individual biological and behavioral factors, and the environmental contexts in which they operate. Child psychology has recently embraced Sameroff and Chandler's Transactional Developmental Model (Sameroff & Chandler, 1975). Transactional models of development see the organism as an active participant in its own growth and differentiation. The constants in development are not the set of traits the organism possesses at any one time, but rather the processes by which these traits are maintained or not maintained by the transactions between the organism and its environment. The transactional model also recognizes the self-righting tendencies in development described by Waddington (1957) and recently applied to infant development by Kagan (1982); it allows for the fact that constitutionally robust children frequently adjust to biological or environmental challenges, except under unusually adverse conditions.

There are numerous instances in the behavioral toxicology and teratology literature where the adoption of a transactional model would have broadened our knowledge about ways in which toxic agents affect human development (Pearson & Dietrich, 1983). However, for the sake of brevity we will focus on only one example which comes from the literature on the behavioral effects of in-utero exposure to psychoactive drugs (heroine and methadone).

Few studies have been done following addicted infants into later infancy and early childhood, and what findings there are on such subjects are equivocal. Yet, there seems to be consensus in the literature as to the withdrawal signs seen in many neonates and infants with narcotics abstinence syndrome (e.g., increased irritability and tremulousness, irregular sleep cycles, and decreased alertness and cuddliness--Householder, Hatcher, Burns, & Chasnoff, 1982). Such signs have also been noted in cases of fetal alcohol syndrome (Rosett & Sander, 1979). In either case, the use of a transactional model would open suppositional doors as to the processes which may lead to developmental delays.

As Figure 4 illustrates, some investigators have recognized that many of the symptoms of withdrawal seen in these infants would severely tax the patience and nurturing skills of a mother. They have proposed that such behaviors on the infant's part could undermine the mother-infant relationship to a degree where, over time, the resulting negative interactions could foster social and/or intellectual delays in the child. The critical point here is that the sequence of events which might lead to such a developmental outcome could only be uncovered through a transactional developmental framework where such relationships are accounted for in the analyses. Though complex, the statistical tools for uncovering such processes are available (e.g., hierarchical step-wise multiple regression, cross-panel correlation, and structural equation approaches such as critical path analysis).

We are currently invloved in a large longitudinal investigation of the neurobehavioral effects of low to moderate lead toxicity in infants and children. Our test battery (shown in Table 1) was formulated so that we might trace the developmental processes which lead from exposure to Pb to the developmental delays which our hypotheses anticipate. There are five aspects of this battery which make it uniquely capable of addressing issues generated by the transactional model as applied to child behavioral toxicology:

1.) The processes involved in determining an infant's developmental trajectory are recognized as starting with events before, during and just after birth (note perinatal risk assessments).

2.) The initial neurological intactness of the infant is recognized as having a potential impact on early social and non-social interactions with the environment, and thus later development (note Brazelton scale, three and six month Bayley's and infant temperament assessments).

3.) The battery doesn't settle for indirect, status oriented measures of the child's developmental environment. Environmental process variables are assessed directly (note Home Observation for Measurement of the Environment and laboratory caretaker-child interaction assessments)

4.) The measures of behavioral development are broadbased. The battery combines both standard psychometric instruments with specific measures of neurobehavioral functioning. Such fine grain measures of neurological status may be more sensitive to the subtle delays one is likely to uncover at low toxicant dosage levels (note visual habituation assessments, discrimination learning, and activity level).

5.) The level of intoxication is assessed at frequent and regular intervals and concurrently with each neurobehavioral evaluation. This will allow the evaluation of both concurrent and latent effects of the toxicant on social and non-social interactions and development.

The transactional developmental perspective in behavioral toxicology and teratology places greater demands on the investigator in terms of research design, data collection, data analysis, and interpretation. However, the issues we are investigating are too important to demand less of ourselves as scientists.

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FIGURE 1

MAIN EFFECT MODEL

BIOLOGICAL AND ENVIRONMENTAL VARIABLES EXERT INFLUENCES ON DEVELOPMENT WHICH ARE INDEPENDENT OF EACH OTHER

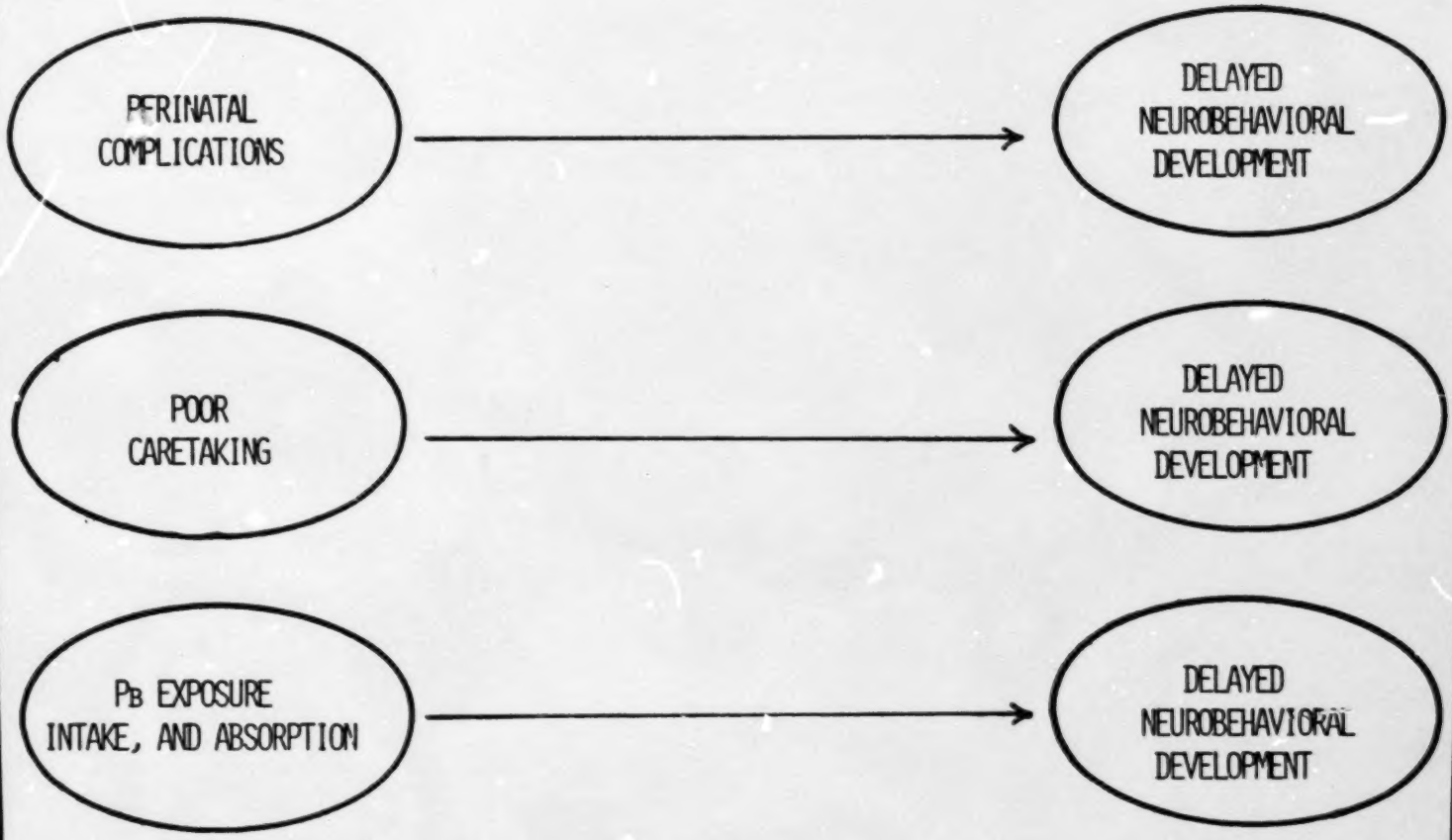

FIGURE 2

FREQUENCY DISTRIBUTION OF TOTAL H.O.M.E. SCORES

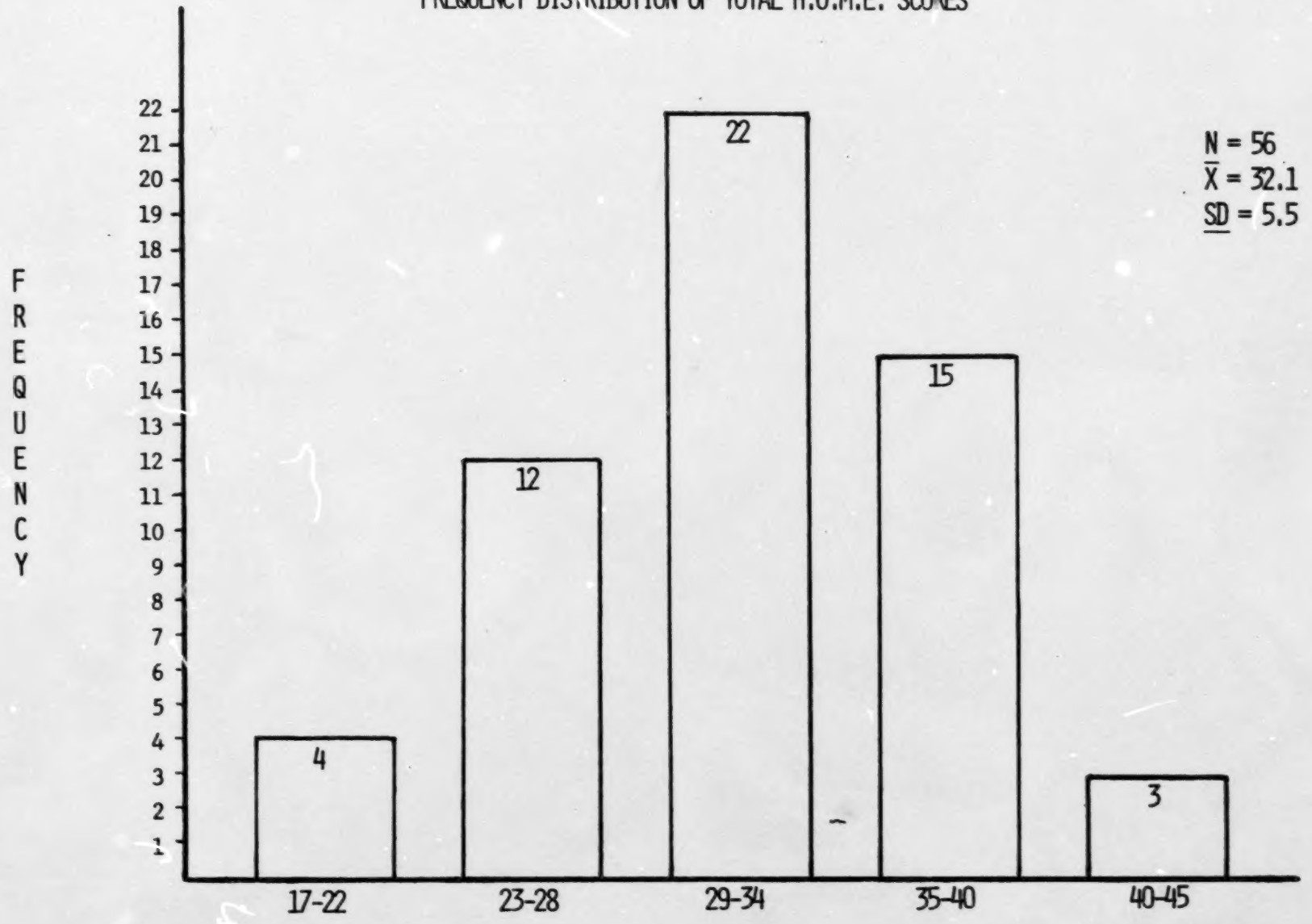

FIGURE 3

STATIC INTERACTIVE MODEL

BIOLOGICAL AND ENVIRONMENTAL VARIABLES EXERT CUMULATIVE DELETERIOUS, POSITIVE, AND AMELIORATING INFLUENCES ON DEVELOPMENT

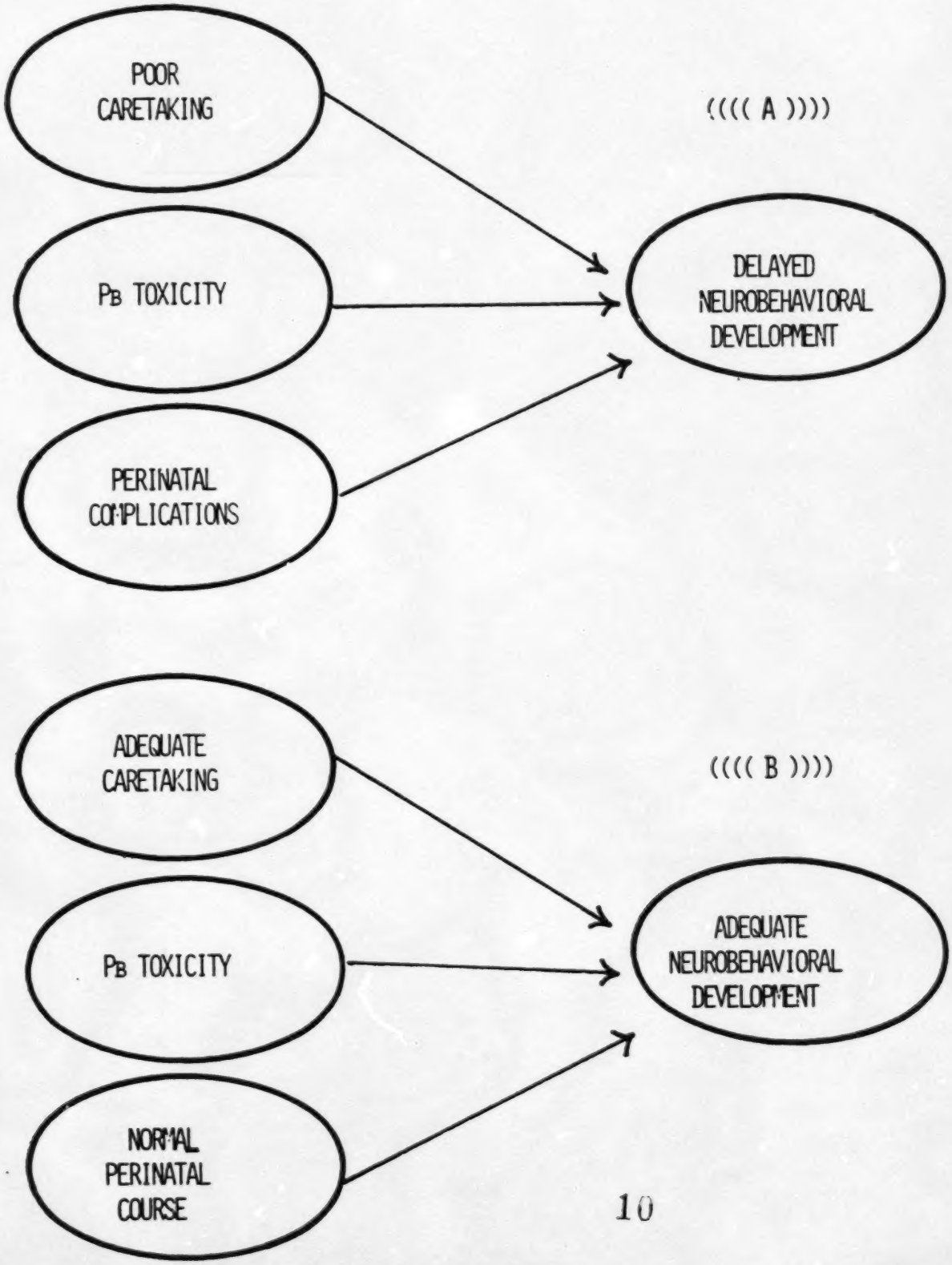

FIGURE 4

TRANSACTIONAL DEVELOPMENTAL PERSPECTIVE: BEHAVIORAL TERATOGENICITY OF PSYCHOACTIVE DRUGS

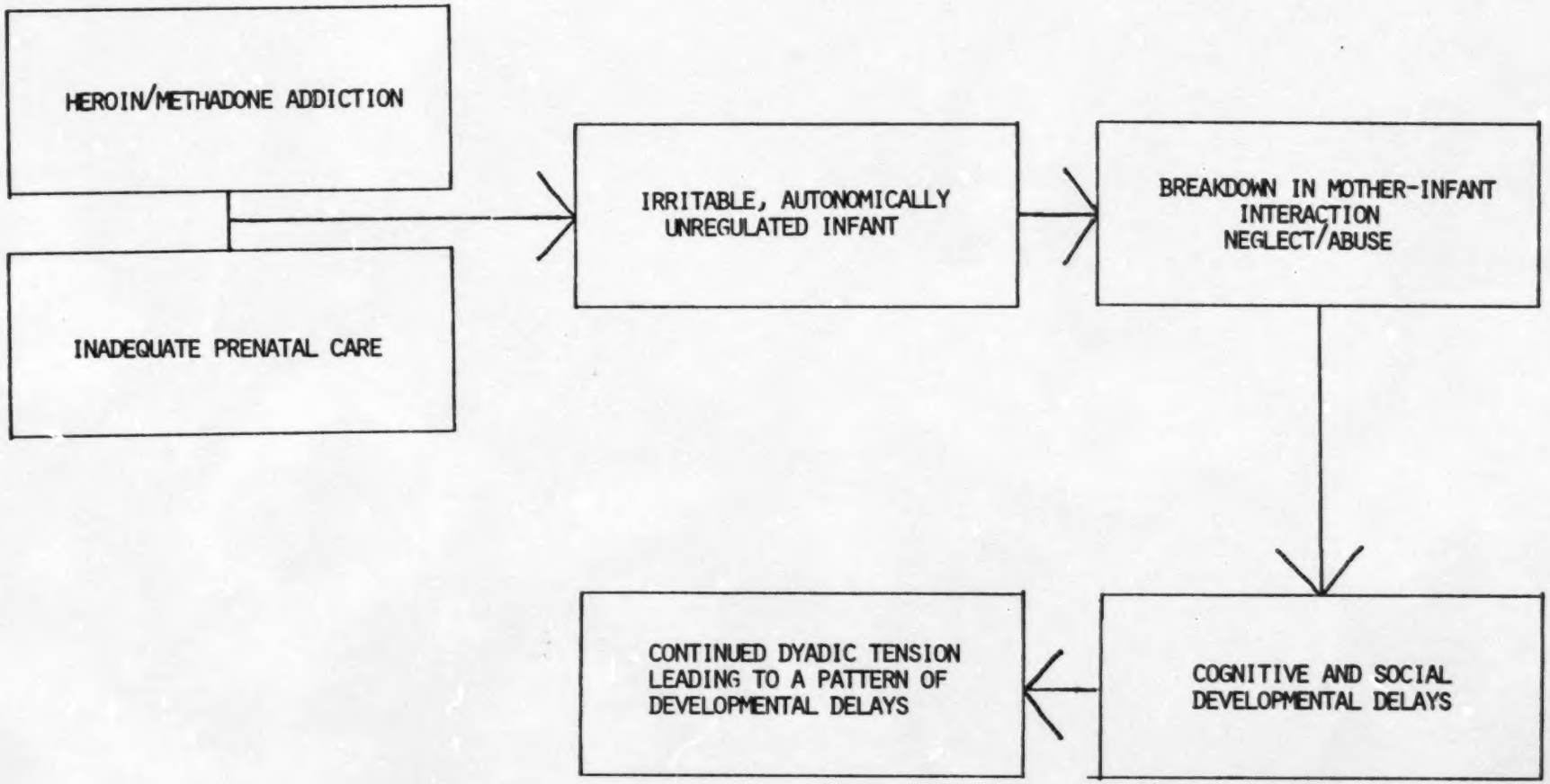
HEROIN/METHADONE ADDICTION
INADEQUATE PRENATAL CARE
IRRITABLE, AUTONOMICALLY UNREGULATED INFANT
BREAKDOWN IN MOTHER-INFANT INTERACTION NEGLECT/ABUSE
COGNITIVE AND SOCIAL DEVELOPMENTAL DELAYS
CONTINUED DYADIC TENSION LEADING TO A PATTERN OF DEVELOPMENTAL DELAYS

TABLE 1

CINCINNATI LEAD PROGRAM PROJECT
BEHAVIORAL TEST BATTERY FOR INFANTS AND YOUNG CHILDREN

I. Perinatal Risk Factors
- Hobel Prenatal Obstetrical and Perinatal Risk Assessment Scale (Birth)
- Littman-Parmelee Obstetrical Risk Assessment (Birth)

II. Postnatal Neurological Status
- Brazelton Newborn Assessment Scale (Birth and 10 Days)
- Bayley Scales of Infant Development (3 Months)

III. Process-Oriented Measures of the Child's Developmental Environment
- Home Observation for Measurement of the Environment (6, 12, 24, 36, 48 and 60 Months)
- Life Events Inventory (5, 12, 24, 36, 48 and 60 Months)

IV. Psychometric Indices of Development
- Bayley Scales of Infant Development (6, 12 and 24 Months)
- McCarthy Scales of Children's Ability (36, 48 and 60 Months)
- Peabody Picture Vocabulary Test (36, 48 and 60 Months)
- Fluharty Preschool Speech and Language Screening Test (36, 48 and 60 Months)

V. Fine-Grain Measures of Behavioral Development
- Habituation (9, 18, 27, 36, 48 and 60 Months)
- Discrimination Learning (18, 27, 36, 48 and 60 Months)
- Activity level (18, 27, 36, 48 and 60 Months)

VI. Reactive Characteristics of the Child
- Pederson Infant Temperament (6 and 12 Months)
- Child Characteristics Questionnaire (24 Months)

VII. Hematological Assessments
- e.g. Blood Lead, Hemoglobin, Hematocrit (Cord Blood and Every 3 Months Until Child is 5 Years of Age)